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HAEMOPHILUS INFLUENZAE TYPE B DISEASE AT 11 PEDIATRIC CENTRES, 1996-1997 . . . . . F-1	105-108	**Official page numbers:** For reference purposes, citing should refer to the page numbers of the printed copy and not to those of the FAX copy (F-#).
VARICELLA-RELATED DEATHS AMONG CHILDREN — UNITED STATES, 1997 . . . . . F-3	108-111	
THE 50TH ANNIVERSARY OF WHO. F-5	111-112	

HAEMOPHILUS INFLUENZAE TYPE B DISEASE AT 11 PEDIATRIC CENTRES, 1996-1997

Introduction

Prior to the introduction of vaccines, *Haemophilus influenzae* type b (Hib) was the most important bacterial pathogen in early childhood, responsible for the majority of cases of meningitis and epiglottitis and contributing to the burden of cases of bacteremia, septic arthritis, pneumonia, and pericarditis [1]. Beginning in 1992, all provinces and territories routinely immunized infants ≥ 2 months of age with the newer, more effective PRP-conjugate vaccines. The Canadian Pediatric Society's Immunization Monitoring Program, Active (IMPACT) previously documented the remarkable decline in Hib cases at participating centres through 1995[2]. This report examines cases of Hib infection seen at IMPACT centres in 1996 and 1997.

Methods

At each of the 11 participating centres, children admitted with Hib infection were identified by reviewing microbiology laboratory and health records[2]. The following information on each child was collected: age, sex, reporting hospital, admission date, manifestations of illness, results of bacterial cultures and antigen tests, level(s) of care required, duration of admission, condition at discharge, and prior Hib vaccination record including dates and names of the products.

Hib infection was classified as definite if the organism was isolated from a normally sterile body fluid, or as probable if Hib antigen was detected in a sample of cerebrospinal fluid, urine, or other normally sterile body fluid. Vaccine failure was defined as the onset of culture-confirmed Hib infection more than 28 days after completion of age-appropriate vaccination.

Results

During 1996 and 1997, 16 cases of Hib infection were reported, eight in each year (Table 1). Fifteen cases were culture-confirmed (definite). Two centres had no cases at all, and four had no cases in 1997. Affected children ranged in age from 2 months to 12 years, with a mean of 55 months. Seven (44%) were > 60 months of age, beyond the expected age for susceptibility to Hib infection. Eleven (69%) were male and five (31%) were female. Fifteen of the 16 cases were considered healthy prior to the onset of Hib infection.

Disease manifestations were most commonly meningitis or epiglottitis, although other syndromes also occurred (Table 2). All but one of the 16 cases were treated in hospital and the majority (63%) required intensive care. Each case survived. At least two of the eight meningitis cases suffered hearing impairment.

In 1996, only one of the eight cases was preventable. This occurred in a 5-year-old male whose single Hib vaccination at 18 months of age was overlooked by his vaccine providers. Two cases were too old (10 and 11 years of age) to have been included in earlier programs. One child was too young (2 months of age) to have completed the primary series. Four had completed age-appropriate vaccination, one with polysaccharide vaccine, two with PRP-D (ProHIBiT®) vaccine, and one with PRP-T (Act-HIB™) vaccine. The latter child had received four doses, including a booster at 18 months of age, prior to developing pneumonia with bacteremia at 3 years of age.

Again in 1997, only one of the eight cases was preventable. In this instance the parents had refused all immunizations; the child developed Hib meningitis at 12 months of age and suffered hearing impairment. Two were too young (2 and 3 months of age) to have

TABLE 1
Number of cases of *Haemophilus influenzae* type b infection, 1996 and 1997, reported by the 11 IMPACT centres

Centre	1996	1997	Total
St. John's	1	0	1
Halifax	1	1	2
Quebec	0	0	0
Montreal (Montreal Children's Hospital)	1	0	1
Montreal (*Hôpital Sainte-Justine pour les enfants*)	2	1	3
Ottawa	1	1	2
Toronto	0	0	0
Winnipeg	1	1	2
Calgary	0	1	1
Edmonton	0	2	2
Vancouver	1	1	2

TABLE 2
Syndromes due to *Haemophilus influenzae* type b during 1996-1997

Syndrome	Number of cases*
Meningitis	8 (50%)
Epiglottitis	5 (31%)
Other (pneumonia, cellulitis, otitis media, bacteremia, pericarditis, etc.)	7

* ≥ 1 syndrome per case

completed primary immunization. Five had completed age-appropriate immunization, one with PRP-D and the others with PRP-T. The latter cases included one with acute immunosuppression for bone marrow transplantation to correct a metabolic disorder. Two failures occurred after three doses of PRP-T and one after four doses in apparently normal children.

Discussion

Eight cases of Hib infection were encountered by IMPACT centres in each of 1996 and 1997. This represents a dramatic decrease from the 485 cases documented in 1985 just prior to the licensure of the first Hib vaccine and a further improvement over the 20 cases reported in 1995[2] despite the subsequent addition of an eleventh surveillance centre.

These 16 recent cases spanned a wide age range, from 2 months to 12 years. Almost one-half of the cases occurred after 60 months of age, beyond the age when Hib disease was considered a significant risk[1]. Those older children had either not been immunized or had received less effective Hib vaccines no longer in use.

The Hib illnesses encountered were severe, with meningitis accounting for 50% of cases and epiglottitis for 31% (Table 2), comparable to past series[1,2]. No deaths were reported. At least two meningitis cases were complicated by hearing impairment.

In contrast to the situation in 1994, when eight of the 24 cases encountered were preventable[3], only two of the cases encountered in 1996 and 1997 were preventable. They included a child whose parents had refused immunizations and another whose single Hib immunization at 18 months had been missed when other vaccines were administered. Among the non-preventable cases were two who were too old to have been included in early programs and three who were too young to have completed primary immunization.

The majority of non-preventable cases (nine) occurred in children who had completed age-appropriate Hib vaccination. Four cases involved vaccines no longer in use, a phenomenon that is becoming less frequent with time. Five cases involved PRP-T vaccine, a remarkably small number considering the widespread use of this vaccine in Canada since 1992. Of these, one case was severely immunocompromised, but the others were apparently healthy children. Of particular note are two cases that occurred following completion of a four-dose series (including the 18-month booster), a phenomenon not previously encountered by IMPACT centres or reported in Canada. Immunologic investigations of these children will be reported separately but both were considered healthy prior to their Hib illness.

In summary, annual Hib case totals at IMPACT centres may be nearing an irreducible minimum. Cases were rare and sporadic: some large centres had no cases in 1996 and 1997, while others encountered Hib anew after 1 to 2 years without cases.The organism continues to attack children at risk. Concerted efforts remain warranted to immunize every eligible child. Such efforts must include effective parent counselling and diligent record-keeping[4]. Judging from the few failures encountered after PRP-T vaccine, its effectiveness must be high.

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Source: *S Grewal, Department of Pediatrics, The University of British Columbia, Vaccine Evaluation Center, British Columbia's Children's Hospital, D Scheifele, MD, Vaccine Evaluation Center, British Columbia's Children's Hospital, Vancouver, BC* .

International Notes

VARICELLA-RELATED DEATHS AMONG CHILDREN — UNITED STATES, 1997

During the first quarter of 1998, the Texas Department of Health and the Iowa Department of Public Health notified the United States Centers for Disease Control and Prevention of three fatal cases of varicella (chickenpox) that occurred in children during 1997. All three children were unvaccinated. Two children contracted chickenpox from unvaccinated siblings, and the mode of exposure was unknown for the third. This report summarizes these cases and indicates that varicella-related deaths continue to occur among children in the United States despite the availability of vaccine and recommendations for its use in all susceptible children[1,2].

Case 1

On 28 February 1997, a previously healthy, unvaccinated 21-month-old boy developed a typical varicella rash. He had no reported exposure to varicella. On 1 March, he was taken to a local emergency department (ED) with a high fever and was started on oral acetaminophen and diphenhydramine. On 3 March, his primary-care physician prescribed oral acyclovir. On 4 March, his mother noted a new petechial-like rash. The next morning, his primary-care physician noted lethargy, a purpuric rash, and poor perfusion. He was transferred to a local ED. Fluid resuscitation and intravenous ceftriaxone were initiated, but the child continued to deteriorate rapidly, requiring intubation, mechanical ventilation, and inotropic support with dopamine. Blood cultures were negative for bacterial pathogens. Laboratory tests indicated disseminated intravascular coagulation and severe dehydration. Approximately 1½ hours after arrival at the ED, he was transported to a tertiary-care centre. Within 10 minutes of arrival, he suffered cardiac arrest and died. The death was attributed to varicella with hemorrhagic complications.

Case 2

On 21 December 1997, a 5-year-old unvaccinated boy with a history of asthma was taken to a local ED with a fever of 40.3° C and a typical varicella rash in multiple stages of healing. The child was treated with antipyretic and antipruritic medications, and discharged.

That evening, the boy developed mild dyspnea and was treated at home for a presumed asthma attack with metered-dose inhalers and one dose of oral prednisone. He returned to the ED on 22 December with shortness of breath and a 4-hour history of abdominal and leg pain. On presentation to the ED, one of the patient's siblings had active varicella and another had recently recovered from varicella. Physicial examination revealed numerous chickenpox lesions, one of which appeared infected. He was tachypneic, and his extremities were mottled consistent with peripheral septic emboli. Chest and abdominal radiographs revealed a right pleural effusion, pneumonia, and mild ileus. Thoracostomy produced pleural fluid containing gram-positive cocci, confirmed 8 hours later to be group A *Streptococcus* (GAS). A peripheral blood sample revealed gram-positive cocci. He was admitted to the hospital and treated with intravenous ceftriaxone, nafcillin, and acyclovir.

After admission, his breathing became labored and his extremities increasingly mottled. He rapidly developed hypotension, obtundation, and bradycardia. Despite efforts at cardiopulmonary resuscitation, the child died 5 hours after arriving at the ED. A post-mortem examination attributed the death to GAS septicemia, pneumonia, and pleural effusion, complicating varicella infection.

Case 3

On 14 December 1996, a previously healthy, unvaccinated 23-month-old boy developed fever and a typical varicella rash. Approximately 1 to 2 weeks earlier, his unvaccinated 4-year-old sibling had contracted varicella. He was taken to his physician on 17 December because of persistent fever and cellulitis of the left foot, and he was hospitalized on 19 December for failure to improve on an unspecified outpatient antibiotic regimen. Because his condition deteriorated despite intravenous methicillin and ceftriaxone, he was transferred to a regional hospital on 21 December. Sepsis, possible viral meningoencephalitis, and mild pleural effusion were diagnosed. A cerebrospinal fluid examination revealed lymphocytic pleocytosis, and blood and urine cultures grew penicillin-resistant *Staphylococcus aureus*. Antibiotics were changed to nafcillin and gentamycin, and intravenous acyclovir was added on 23 December. On 24 December, the child developed an aortic insufficiency murmur, and an echocardiogram revealed a 9 × 9 mm vegetation on the

aortic valve, consistent with bacterial endocarditis. Serial echocardiograms displayed growth of the vegetation and development of a pericardial effusion. He was transferred to a cardiac surgery centre on 26 December. While awaiting surgery, he developed refractive heart failure secondary to staphylococcal endocarditis. He became incoherent, probably secondary to a major embolic neurologic event, and died on 8 January 1997.

MMWR Editorial Note

The three cases described in this report indicate that healthy children continue to die from complications of varicella, a disease that is preventable through vaccination. Although commonly viewed as a benign disease of childhood, serious complications and death can occur following varicella. Varicella is the leading cause of vaccine-preventable deaths in children in the United States.

During 1990-1994, varicella was the underlying cause of death in an average of 43 children aged < 15 years each year (CDC, unpublished data, 1998). During 1988-1995, up to 10,000 children were hospitalized each year for varicella or its complications (CDC, unpublished data, 1998). Ninety percent of the children who died did not have high-risk conditions for severe varicella. The most common severe complications from varicella among fatal cases in children are secondary bacterial infections and pneumonia. Other complications include encephalitis, hemorrhagic complications, hepatitis, arthritis, and Reye syndrome. Reports of severe invasive infections from GAS-complicating varicella have heightened awareness that varicella is a well-defined risk factor for GAS disease[3,4].

Varicella vaccine was licensed in the United States in March 1995, is widely available, and is recommended for routine vaccination of children aged 12 to 18 months and for vaccination of susceptible older children, adolescents, and adults[1,2]. The Vaccines For Children (VFC) program provides varicella vaccine for VFC-eligible children aged > 12 months who were born on or after 1 January 1983, and for VFC-eligible children aged < 19 years who are family members of an immunocompromised person.

National coverage levels among children aged 19 to 35 months for varicella vaccine have increased from 14% during July to September 1996 to 25% during March to June 1997[5]. Barriers to vaccine use include the perception that varicella is a benign disease, concerns that immunity will not persist, the potential that varicella disease burden will shift to older age groups among whom the disease is more severe, and concerns about vaccine efficacy and safety[4]. A recent study documented 100% vaccine efficacy for prevention of moderate or severe varicella and 86% for prevention of all varicella[6]. In addition, vaccinated children who developed varicella caused by wild virus or "breakthrough disease" had very mild disease of short duration with < 50 lesions[7]. Persistence of immunity for more than 20 years post-vaccination has been demonstrated[8]. As disease incidence and exposure to wild virus declines, continuing surveillance will determine the need for and timing of additional doses of vaccine.

To monitor the impact of varicella vaccination programs throughout the United States, varicella surveillance is needed; surveillance for varicella deaths in all states is a key first step in this process. States also are encouraged to develop additional sustainable surveillance systems, including monitoring hospitalizations and establishing statewide aggregate reporting for cases by schools, day-care centres, and/or health-care provider offices, and to consider instituting vaccine requirements for day care and school entry[1].

Efforts to increase routine and catch-up varicella vaccination among children should include educating health-care providers that deaths and severe morbidity from varicella are preventable[1,2]. Policies that delay vaccination of susceptible children until adolescence accept the considerable disease burden that occurs among children aged 2 to 11 years. The most effective vaccination strategy focuses on vaccinating children routinely at age 12 to 18 months, and vaccinating all susceptible older children and adolescents. Children have the highest disease incidence and are the group that serve as the primary source of transmission of varicella to groups at higher risk for severe disease, including adults[9] and persons who are not eligible for vaccination. Most deaths and severe morbidity from varicella in children in adults can be prevented by implementing recommended policies for childhood vaccination.

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Source: *Morbidity and Mortality Weekly Report, Vol 47, No 18, 1998.*

THE 50TH ANNIVERSARY OF WHO

The *World Health Report 1998 – Life in the 21st century: A vision for all*, commemorates WHO's 50th anniversary. The report describes international health events over the past 50 years.

During the past few decades, substantial progress has been made in controlling some major infectious diseases. Some have disappeared or are almost eliminated as public health problems:

- Global eradication of **smallpox** was declared in 1980 at the end of an eradication campaign which began in 1967.
- The tropical disease **yaws**, which mainly affects the skin and bones, has virtually disappeared.
- The global threat of **plague** has declined in the past four decades, largely due to the impact of antibiotics, insecticides, and other control measures, but cyclical epidemics still occur.
- Improvements in sanitation and hygiene standards in recent decades have made outbreaks of **relapsing fever** transmitted by lice rare; they are most likely to occur in unhygienic and crowded conditions arising from wars or natural disasters.
- The **onchocerciasis** control program, which began in several countries of West Africa in 1974, has protected an estimated 36 million people from the disease. The African Programme for Onchocerciasis Control began in January 1996 and covers 19 additional countries. The Onchocerciasis Elimination Programme in the Americas was started in 1991 in six Latin American countries, and aims to eliminate severe pathologic manifestations of the disease and to reduce morbidity. It is expected that the global elimination of onchocerciasis as a public-health problem will be achieved before 2008.
- Since the first effective vaccines against **poliomyelitis** were introduced in 1955, the disease has gradually been eliminated in much of the world, with cases declining by more than 90% since the campaign for global eradication by the year 2000 was launched in 1988. The disease has disappeared from the Americas, and global elimination is a feasible goal.
- WHO developed and promoted a multidrug therary for **leprosy**, which it began to recommend in the 1980s. The global leprosy burden has since been reduced greatly. WHO's goal is to eliminate leprosy as a public health problem by the year 2000.
- Progress towards elimination of **dranunculiasis** (guinea-worm disease) in the past decade has been spectacular, with the number of cases falling dramatically worldwide; it is now confined to 17 countries (16 in sub-Saharan Africa and Yemen).
- The outlook for **filariasis** control and elimination is encouraging, and in 1997 the Health Assembly called for the elimination of lymphatic filariasis as a public-health problem globally.

Alongside the significant progress made, many other communicable diseases remain daunting public-health threats; these will be reported on in a future issue.

Source: *WHO Weekly Epidemiological Record, Vol 73, No 20, 1998.*

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